AMENDMENTS

In the Specification:

Please amend paragraph 24 on page 7 as follows:

Figure 4 illustrates the effect of the LXR pan-agonist Compound 1 on lipoprotein lipase (LPL) mRNA levels in wildtype, LXRα-/-, LXRβ-/-, and LXRαβ-/- mice. Compound 1 (10 mg/kg) was dosed daily for seven days by oral gavage. LPL levels were measured by quantitative PCR of total liver RNA. Data is expressed as fold induction by Compound 1 (+Compound 1/ Vehicle, hatched bars). The value for vehicle treated mice in each group was set at 1.0 (white bars). Data is the average of four animals per group assayed in triplicate. *Signifies that the value is statistically different from the vehicle treated value within each genotype.

Please replace paragraph 25 on page 7 with the following amended paragraph:

Figure 5 illustrates the effect of the LXR pan-agonist Compound 1 on HDL cholesterol levels in wildtype, LXRα-/-, LXRβ-/-, and LXRαβ-/- mice. Compound 1 (10 mg/kg) was dosed daily for seven days by oral gavage. HDL levels were determined from plasma samples taken on day 7. Data presented is the average value derived from seven animals in each group except for $LXR\alpha^{-/-}$/C57BL/6 which is the average of six animals. (+Compound 1/Vehicle, hatched bars; vehicle only, white bars.) *Signifies that the value is statistically different from the vehicle treated value within each genotype.

Please replace paragraph 26 on page 8 with the following amended paragraph:

Figure 6 illustrates the effect of the LXR pan-agonist Compound 1 on CYP7a mRNA levels in wildtype, LXRα-/-, LXRβ-/-, and LXRαβ-/- mice. Compound 1 (10 mg/kg) was dosed daily for seven days by oral gavage. CYP7a levels were measured by quantitative PCR of total liver RNA. Data is expressed as fold induction by Compound 1 (+Compound 1 /Vehicle, hatched bars). The value for vehicle treated mice in each group was set at 1.0 (white bars). Data

is the average of four animals per group assayed in triplicate. *Signifies that the value is statistically different from the vehicle treated value within each genotype.

Please replace paragraph 27 on page 8 with the following amended paragraph:

Figure 7 illustrates the effect of the LXR pan-agonist Compound 1 on dietary cholesterol absorption. Compound 1 (50 mg/kg) was dosed daily for seven days by oral gavage. Cholesterol absorption was than measured using the fecal extraction method. Data is expressed as the percentage of radiolabeled cholesterol that was absorbed and is the average of seven animals in each group. (+Compound 1/Vehicle, hatched bars; vehicle only, white bars.) *Signifies that the value is statistically different from the vehicle treated control value.

Please replace paragraph 28 on page 8 with the following amended paragraph:

Figure 8 illustrates the effect of the LXR pan-agonist Compound 1 on ABCA1 mRNA levels in the intestines of wildtype, LXRα-/-, LXRβ-/-, and LXRαβ-/- mice. Compound 1 (10 mg/kg) was dosed daily for seven days by oral gavage. ABCA1 levels were measured by quantitative PCR of total intestinal mucosa RNA. Data is expressed as fold induction by Compound 1 (+Compound 1/Vehicle, hatched bars). The value for vehicle treated mice in each group was set at 1.0 (white bars). Data is the average of four animals per group assayed in triplicate. *Signifies that the value is statistically different from the vehicle treated value within each genotype.

Please replace paragraph 29 on page 8 with the following amended paragraph:

Figure 9A shows representative sudan IV stained *en face* aorta preparations from ApoE-/- mice following LXRαβ-/- bone marrow transplants. Atherosclerotic lesions stain red. Figure 9B illustrates the effects of LXRαβ-/- bone marrow transplants on ApoE-/- mice via quantitation of the surface area of aortas covered with lesions. Data is the average of six aortas for the ApoE-/- toApoE-/- group and seven aortas for the wildtype to ApoE-/- and LXRαβ-/- to ApoE-/- groups.

*Signifies that the value is statistically different from the ApoE-/- to ApoE-/- control bone marrow transplant value.

Please replace paragraph 30, at page 9 with the following amended paragraph:

Figure 10A shows representative sudan IV stained *en face* aorta preparations from LDLR-/- mice following LXRαβ-/- bone marrow transplants. Atherosclerotic lesions stain red. Figure 10B illustrates the effects of LXRαβ-/- bone marrow transplants on LDLR-/- mice via quantitation of the surface area of aortas covered with lesions. Data is the average of seven aortas for the LDLR-/- to LDLR-/- group, 11 aortas in the wildtype to LDLR-/- group, and 12 aortas in the LXRαβ-/- to LDLR-/- group. *Signifies that the value is statistically different from the LDLR-/- to LDLR-/- control bone marrow transplant value.

Please replace paragraph 32 at page 9 with the following amended paragraph:

Figure 12A illustrates the effect of the LXR pan-agonist Compound 1 on ABCA1 mRNA levels in peritoneal macrophages isolated from mixed wildtype, LXRα-/- (C57BL/6), LXRβ-/-, and LXRαβ-/- mice. Peritoneal macrophages were cultured *in vitro* for 24 hours in the absence (white bars) or presence (hatched bars) of 1.0 μM Compound 1, total RNA was isolated and the levels of the ABCA1 mRNA were determined by quantitative PCR. Values reported are the averages of three samples for each group assayed in triplicate. Numbers above the hatched bars are the values for the fold induction by Compound 1 (+Compound 1/Vehicle). Figure 12B illustrates the effect of the LXR pan-agonist Compound 1 on ABCG1 mRNA levels in peritoneal macrophages isolated from mixed wildtype, LXRα-/- (C57BL/6), LXRβ-/-, and LXRαβ-/- mice. Peritoneal macrophages were cultured *in vitro* for 24 hours in the absence (white bars) or presence (hatched bars) of 1.0 μM Compound 1, total RNA was isolated and the levels of the ABCG1 mRNA were determined by quantitative PCR. Values reported are the averages of three samples for each group assayed in triplicate. Numbers above the hatched bars are the values for the fold induction by Compound 1 (+Compound 1/Vehicle).